Delayed paraplegia 10 months after endovascular repair of thoracic aortic aneurysm

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Spinal cord ischemia after treatment of thoracic pathologies remains a devastating problem. A 74-year-old man with a history of infrarenal abdominal aortic aneurysm repair presented with bilateral common iliac and left femoral aneurysms as well as a thoracic aortic aneurysm. He underwent an open repair of the iliac and femoral aneurysms, followed by thoracic endovascular aneurysm repair in a staged manner without complications. Ten months later, he presented with hypotension, and permanent paraplegia developed. (J Vasc Surg 2008;47:625-8.)

Endovascular repair of thoracic aortic aneurysm has gained a wide popularity as an alternative treatment of thoracic aortic aneurysm owing to its minimally invasive nature and lower attendant risk of paraplegia.[1] The risk of this most feared complication is the highest during the 30-day perioperative period and is often associated with hypotension. An unusual case of delayed paraplegia that developed 10 months after a successful thoracic endovascular aortic aneurysm repair (TEVAR) is presented.

CASE REPORT

A 74-year-old man presented with 8-cm thoracic aortic, 5-cm bilateral common iliac, and 2-cm left common femoral artery aneurysms (Fig 1). He had undergone an open infrarenal abdominal aortic aneurysm repair with a tube graft 11 years earlier. A decision was made to repair the bilateral common iliac artery aneurysms, followed by TEVAR, in a staged fashion. On June 27, 2005, he underwent aorta-right common iliac/left common femoral bypass grafting with a left hypogastric artery bypass graft to minimize the risk of paraplegia when the thoracic aneurysm was repaired later; the right hypogastric artery was ligated (Fig 2). All graft-vessel anastomoses were done in an end-to-end fashion. The operation was uneventful, and the patient discharged to home on postoperative day 5.

He returned on July 13, 2005, and underwent a TEVAR with adjunctive spinal fluid drainage (Fig 3). Access was obtained through the left limb of the aortic graft. Three TAG (W. L. Gore & Assoc, Flagstaff, Ariz) thoracic endoprostheses (40 mm × 15 cm, 37 mm × 20 cm, and 34 mm × 20 cm) were used. The entire thoracic aorta, from the left subclavian artery origin to the celiac axis/superior mesenteric artery common trunk, was covered. The patient tolerated the procedure well, without any complications, and was released to home the next day.

The patient was seen in the office for a 1-month postoperative follow-up visit. He was doing well, without any neurologic deficits of the lower extremities. A computed tomography (CT) scan showed a type II endoleak from an intercostal artery in the distal thoracic aorta near the landing zone. No type I endoleak was visualized. The left hypogastric bypass graft was patent with cross-filling of the right hypogastric artery (Fig 4). The patient was instructed to have a follow-up CT in 6 months. A follow-up CT was obtained at another hospital in January 2006 and showed a stable aneurysm with no significant change in size and a persistent type II endoleak. The hypogastric bypass graft was noted to be patent. The plan was to study him again in 6 months.

On May 26, 2006, he presented to a local hospital emergency department with fever, abdominal pain, and diarrhea for 5 days. He complained of bilateral lower extremity motor loss in an ambulance ride to the hospital. At the initial evaluation, his blood pressure was 90/50 mm Hg and his neurologic examination was unremarkable. He was noted to have palpable bilateral pedal pulses. Admission laboratory studies were unremarkable.

He was admitted with a diagnosis of diverticulitis, and therapy with intravenous antibiotics was begun. He remained hypotensive. Several hours after admission, bilateral lower extremity numbness developed, followed by loss of sensation in the lower half of the body. Complete paraplegia developed ≤18 hours of admission.

A magnetic resonance image of the thoracic spine was obtained on May 30, 2006 and showed no abnormalities; unfortunately, the lumbar area was not studied, although it is not a foolproof study for detection of spinal cord ischemia (SCI). The patient was thought to have suffered a spinal cord infarction, and no further interventions, such as cerebrospinal fluid (CSF) drainage, were rendered. He was discharged to a rehabilitation center 10 days later.

The left hypogastric bypass graft remains patent as of March 2, 2007 (Fig 5). The patient remains paraplegic, confined to bed, and sacral decubitus ulcers have since developed. He has undergone a

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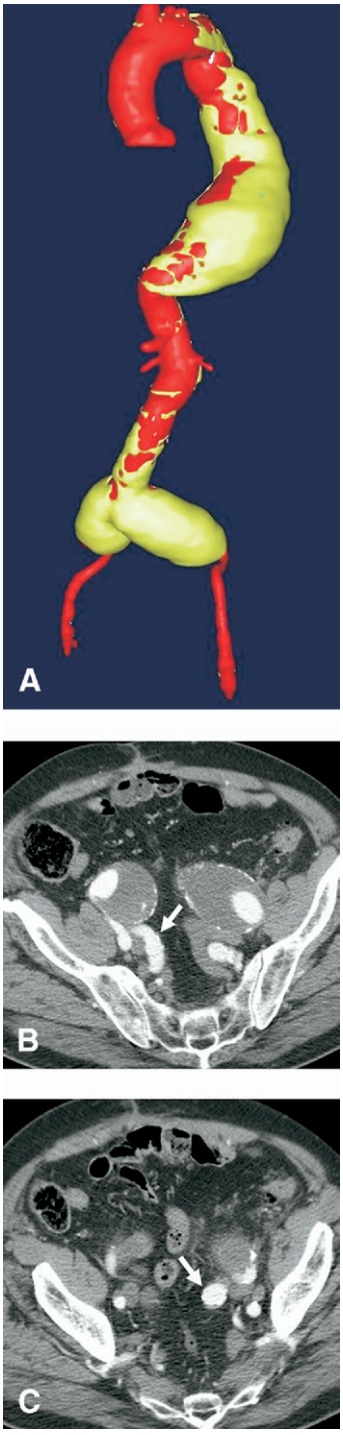

Fig 1. Panel A, A three-dimensional reconstruction of thoracic and bilateral iliac artery aneurysms. *Arrows* indicate **(B)** patent right hypogastric and **(C)** left hypogastric arteries. He had undergone an open repair of infrarenal aortic aneurysm in the past.

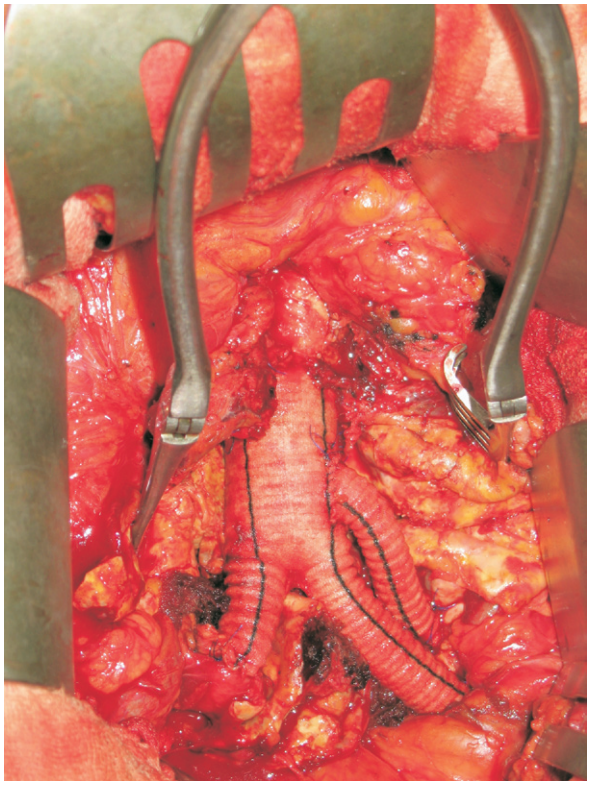

Fig 2. An intraoperative photograph shows left hypogastric artery bypass grafting.

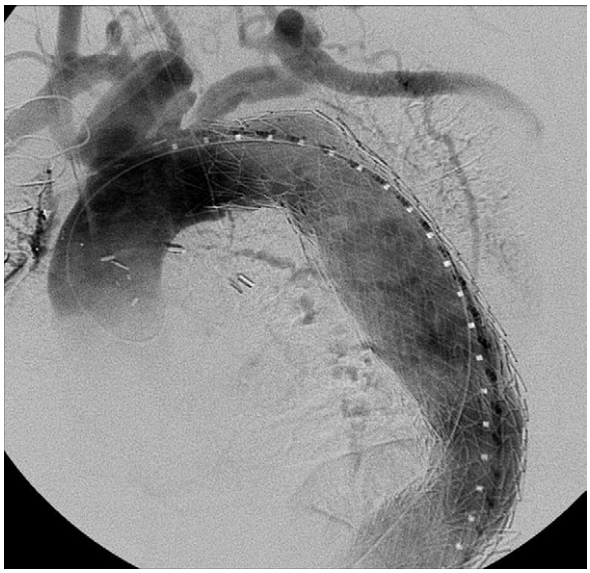

Fig 3. Stent grafting of the thoracic aortic aneurysm with a TAG endoprostheses (W. L. Gore & Assoc, Flagstaff, Ariz).

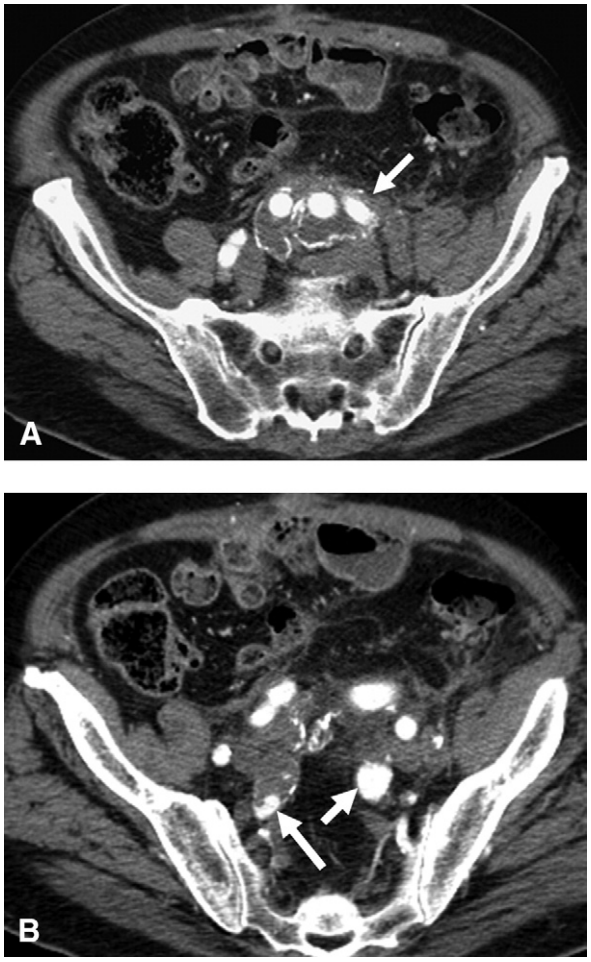

Fig 4. A postoperative computed tomography scan at 1 month shows **(A)** patent left hypogastric artery bypass graft *(arrow)* and **(B)** patent left distal hypogastric artery *(short arrow)* with cross filling of the right *(long arrow)*.

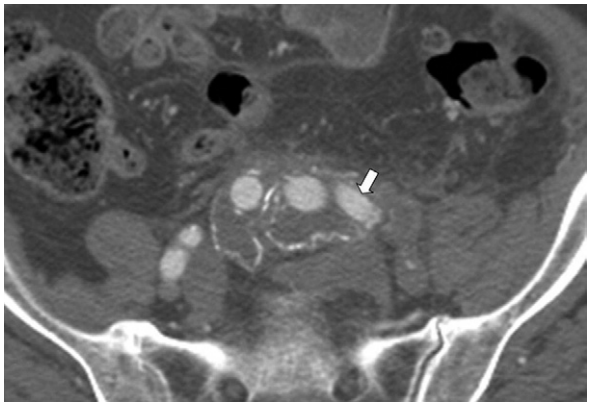

Fig 5. A postoperative computed tomography scan at 10 months shows the patent left hypogastric artery bypass graft *(arrow)*.

diverting colostomy to help heal the wound. The thoracic aneurysm remains stable in size with persistent type II endoleak.

DISCUSSION

Paraplegia or paraparesis occurs as a result of obliteration of blood flow to the spinal cord during thoracic or thoracoabdominal aortic surgery. The obliteration of the branches contributing to the spinal cord blood supply is normally compensated by a collateral network maintaining perfusion of the cord.[2] When this collateral network is tenuous, SCI occurs, either immediate or delayed.

The subject of delayed paraplegia has gained relatively little attention compared with its immediate counterpart. Its incidence after open repairs varies from 0.8% to 13.8% in recent series.[3-11] In some series, the delayed presentation constitutes most of the SCI,[4,9] occurring as late as 91 days postoperatively.[10] A shift in the pattern of paraplegia may occur, with more frequent delayed paraplegia than previously observed.[9] The vulnerable spinal cord perfusion that may have been protected with adjunctive therapy, such as CSF drainage, distal aortic perfusion, naloxone, and intercostal artery reimplatation[8,12-18] may remain susceptible to late development of SCI once these measures are discontinued.[7]

The literature is rather sparse on the late development of SCI after TEVAR. The reported incidence is 2.2% to 12.0%, occurring up to 1.5 years postoperatively.[1,5,19-24] SCI after TEVAR has a number of causes, although precise identification of risk factors is difficult owing to the small number of cases. Several factors have been implicated, including hypotension, prior abdominal aortic aneurysm repair, length of aorta covered, use of an iliac conduit, and coverage of the hypogastric artery and the left subclavian artery.[9,10,19-23] Of these, the first three listed appear to be most important. One additional mechanism that is unique in the pathogenesis of SCI after TEVAR is that coverage of critical intercostals may not necessarily result in immediate paraplegia because flow may be maintained through cross-collateral flow (ie, endoleak). When these endoleaks are sealed, the collateral flow is lost, which then may be followed by the neurologic deficit.[25]

The patient presented in this report harbored most of the risk factors and illustrates that the risk of SCI after TEVAR is not just confined to the perioperative period but may persist for an extended period of time that may be as long as the patient's life span. Hemodynamic compromise to the spinal cord may exacerbate this risk at any time; thus, avoidance of hypotension may be of critical importance in this cohort of patients. Patient education and medical alert tags may be essential in preventing the remote incidence of paraplegia.

It is unclear whether the preservation of bilateral pelvic flow would have prevented the development of SCI in this patient. Contributions of profunda femoral flow through its pudendal and circumflex branches and iliosacral arteries, which are branches of the hypogastric arteries to spinal cord perfusion, have been well illustrated.[26] We believe that if at all possible, all efforts should be made to preserve at least

one, if not both, hypogastric arteries to maximize pelvic and spinal cord perfusion and thereby reduce the risk of paraplegia. The risk of SCI is ongoing, and education of the patients and medical personnel of precautionary measures may be beneficial to avoid this devastating complication.

REFERENCES

1. Cho JS, Haider S, Makaroun MS. US multi-center trials of endoprostheses for the endovascular treatment of descending thoracic aneurysms. J Vasc Surg 2006;43(suppl A):12A-9A.
2. Griepp RB, Griepp EB. Spinal cord perfusion and protection during descending thoracic and thoracoabdominal aortic surgery: the collateral network concept. Ann Thorac Surg 2007;83:S865-9.
3. Cambria RP, Davison JK, Zannetti S, L'Italien G, Atamian S. Thoracoabdominal aneurysm repair: perspectives over a decade with the clamp-and-sew technique. Ann Surg 1997;226:294-303.
4. Cheung AT, Weiss SJ, McGarvey ML, Stecker MM, Hogan MS, Escherich A, et al. Interventions for reversing delayed-onset postoperative paraplegia after thoracic aortic reconstruction. Ann Thorac Surg 2002; 74:413-9.
5. Chiesa R, Melissano G, Marrocco-Trischitta MM, Civilini E, Setacci F. Spinal cord ischemia after elective stent-graft repair of the thoracic aorta. J Vasc Surg 2005;42:11-7.
6. Cina CS, Lagana A, Bruin G, Ricci C, Doobay B, Tittley J, et al. Throacoabdominal aortic aneurysm repair: A prospective cohort study of 121 cases. Ann Vasc Surg 2002;16:631-8.
7. Estrera AL, Miller CC 3rd, Huynh TT, Azizzadeh A, Porat EE, Vinnerkvist A, et al. Preoperative and operative predictors of delayed neurologic deficit following repair of thoracoabdominal aortic aneurysm. J Thorac Cardiovasc Surg 2003;126:1288-94.
8. Jacobs MJ, Mess W, Mochtar B, Nijenhuis RJ, Statius van Eps RG, et al. The value of motor evoked potentials in reducing paraplegia during thoracoabdominal aneurysm repair. J Vasc Surg 2006;43:239-46.
9. Maniar HS, Sundt TM 3rd, Prasad SM, Chu CM, Camillo CJ, Moon MR, et al. Delayed paraplegia after thoracic and thoracoabdominal aneurysm repair: a continuing risk. Ann Thorac Surg 2003;75:113-9.
10. Wong DR, Coselli JS, Amerman K, Bozinovski J, Carter SA, Vaughn WK, et al. Delayed spinal cord deficits after thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 2007;83:1345-55.
11. Go MR, Cho JS, Makaroun MS. Late results of a multicenter study of TEVAR vs. open repair. Perspect Vasc Surg Endovasc Ther 2007;19: 124-30.
12. Safi HJ, Miller CC, III, Huynh TT, Estrera AL. Distal aortic perfusion and cerebrospinal fluid drainage for thoracoabdominal and descending thoracic aortic repair: ten years of organ protection. Ann Surg 2003; 238:372-81.
13. Coselli JS, LeMaire SA, Koksoy C, Schmittling ZC, Curling PE. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: Results of a randomized clinical trial. J Vasc Surg 2002;35:631-9.
14. Safi HJ. Role of the BioMedicus pump and distal aortic perfusion in thoracoabdominal aortic aneurysm repair. Artificial Organs 1996;20: 694-9.
15. Safi HJ, Campbell MP, Miller CC, Iliopoulos DC, Khoynezhad A, Letsou GV, et al. Cerebral spinal fluid drainage and distal aortic perfusion decrease the incidence of neurological deficit: the results of 343 descending and thoracoabdominal aortic aneurysm repairs. Eur J Vasc Endovasc Surg 1997;14:118-24.
16. Acher CW, Wynn MM, Hoch JR, Popic P, Archibald J, Turnipseed WD. Combined use of cerebral spinal fluid drainage and naloxone reduces the risk of paraplegia in thoracoabdominal aneurysm repair. J Vasc Surg 1994;19:236-46.
17. Kunihara T, Matsuzaki K, Shiiya N, Saijo Y, Yasuda K. Naloxone lowers cerebrospinal fluid levels of excitatory amino acids after thoracoabdominal aortic surgery. J Vasc Surg 2004;40:681-90.
18. Safi HJ, Miller CC, Carr C, Iliopoulos DC, Dorsay DA, Baldwin JC. Importance of intercostal artery reattachment during thoracoabdominal aortic aneurysm repair. J Vasc Surg 1998;27:58-68.
19. Gravereaux EC, Faries PL, Burks JA, Latessa V, Spielvogel D, Hollier LH, et al. Risk of spinal cord ischemia after endograft repair of thoracic aortic aneurysms. J Vasc Surg 2001;34:997-1003.
20. Khoynezhad A, Donayre CE, Bui H, Kopchok GE, Walot I, White RA. Risk factors of neurologic deficit after thoracic aortic endografting. Ann Thorac Surg 2007;83:S882-9.
21. Kieffer E, Chiche L, Cormier E, Guegan H. Recurrent spinal cord ischemia after endovascular stent grafting for chronic traumatic aneurysm of the aortic isthmus. J Vasc Surg 2007;45:831-3.
22. Baril DT, Carroccio A, Ellozy SH, Palchik E, Addis MD, Jacobs TS, et al. Endovascular thoracic aortic repair and previous or concomitant abdominal aortic repair: is the increased risk of spinal cord ischemia real? Ann Vasc Surg 2006;20:188-94.
23. Cheung AT, Pochettino A, McGarvey ML, Appoo JJ, Fairman RM, Carpenter JP, et al. Strategies to manage paraplegia risk after endovascular stent repair of descending thoracic aortic aneurysms. Ann Thorac Surg 2005;80:1280-8.
24. Greenberg RK, Resch T, Nyman U, Lindh M, Brunkwall J, Brunkwall P, et al. Endovascular repair of descending thoracic aortic aneurysms: an early experience with intermediate-tem follow-up. J Vasc Surg 2000; 31:147-56.
25. Carroccio A, Marin ML, Ellozy S, Hollier LH. Pathophysiology of paraplegia following endovascular thoracic aortic aneurysm repair. J Cardiac Surg 2001;11:359-66.
26. Connolly JE, Ingegno M, Wilson SE. Preservation of the pelvic circulation during infrarenal aortic surgery. Cardiovasc Surg 1996;4:65-70.

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